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Population-based study of ischemic stroke risk after trauma in children and young adults.

Journal
Neurology, 89(23)

ISSN
0028-3878

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Publication Date
2017-12-01

DOI
10.1212/wnl.0000000000004708

Peer reviewed

Population-based study of ischemic stroke risk after trauma in children and young adults

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ABSTRACT

Objective: To quantify the incidence, timing, and risk of ischemic stroke after trauma in a population-based young cohort.

Methods: We electronically identified trauma patients (<50 years old) from a population enrolled in a Northern Californian integrated health care delivery system (1997–2011). Within this cohort, we identified cases of arterial ischemic stroke within 4 weeks of trauma and 3 controls per case. A physician panel reviewed medical records, confirmed cases, and adjudicated whether the stroke was related to trauma. We calculated the 4-week stroke incidence and estimated stroke odds ratios (OR) by injury location using logistic regression.

Results: From 1,308,009 trauma encounters, we confirmed 52 trauma-related ischemic strokes. The 4-week stroke incidence was 4.0 per 100,000 encounters (95% confidence interval [CI] 3.0–5.2). Trauma was multisystem in 26 (50%). In 19 (37%), the stroke occurred on the day of trauma, and all occurred within 15 days. In 7/28 cases with cerebrovascular angiography at the time of trauma, no abnormalities were detected. In unadjusted analyses, head, neck, chest, back, and abdominal injuries increased stroke risk. Only head (OR 4.1, CI 1.1–14.9) and neck (OR 5.6, CI 1.03–30.9) injuries remained associated with stroke after adjusting for demographics and trauma severity markers (multisystem trauma, motor vehicle collision, arrival by ambulance, intubation).

Conclusions: Stroke risk is elevated for 2 weeks after trauma. Onset is frequently delayed, providing an opportunity for stroke prevention during this period. However, in one-quarter of stroke cases with cerebrovascular angiography at the time of trauma, no vascular abnormality was detected. *Neurology*® 2017;89:2310–2316

GLOSSARY

BCVI = blunt cerebrovascular injury; **CI** = confidence interval; **CTA** = CT angiography; **ICD-9** = *International Classification of Diseases-9*; **KPNC** = Kaiser Permanente Northern California; **OR** = odds ratio.

Ischemic stroke in children and young adults is an important health issue because of the high costs to patients, families, and society. Survivors of childhood stroke may experience years of cognitive or physical disability and poorer health-related quality of life.[1] Approximately a third of young adult stroke patients have poor long-term functional outcomes.[2] An important etiology for ischemic stroke in the young is trauma, often through blunt cerebrovascular injury (BCVI) to cervical or intracranial arteries from forceful cervical rotation, hyperextension, or high-energy collision.[3–5] Trauma-related strokes are important to address because the lifetime costs of disability after stroke in a young person are disproportionately high, and some of these strokes may be preventable.

A clear understanding of the epidemiology of ischemic stroke related to trauma is needed to effectively design strategies to prevent these strokes. However, the epidemiology of ischemic stroke after trauma is still not well-established. Stroke onset may be delayed by hours or days after trauma,[6–8] but the time period at risk has been ill-defined. Because trauma patients often are not systematically followed beyond the initial trauma encounter, strokes that occur

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From the Departments of Neurology (C.K.F., A.L.N., H.J.F.), Pediatrics (C.K.F., A.L.N., H.J.F.), Epidemiology and Biostatistics (N.K.H.), and Surgery (R.A.D.), University of California, San Francisco; the Division of Research (D.R.V., S.S.), Kaiser Permanente Northern California, Oakland; and the Department of Emergency Medicine (D.R.V.), Kaiser Permanente Sacramento Medical Center, Sacramento, CA.

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later could be missed. Dissection-related strokes can occur after minor injuries,[9,10] but patients with minor trauma might not be included in studies if they were seen outside of a trauma center. Finally, little is known about stroke risk in trauma patients without BCVI.

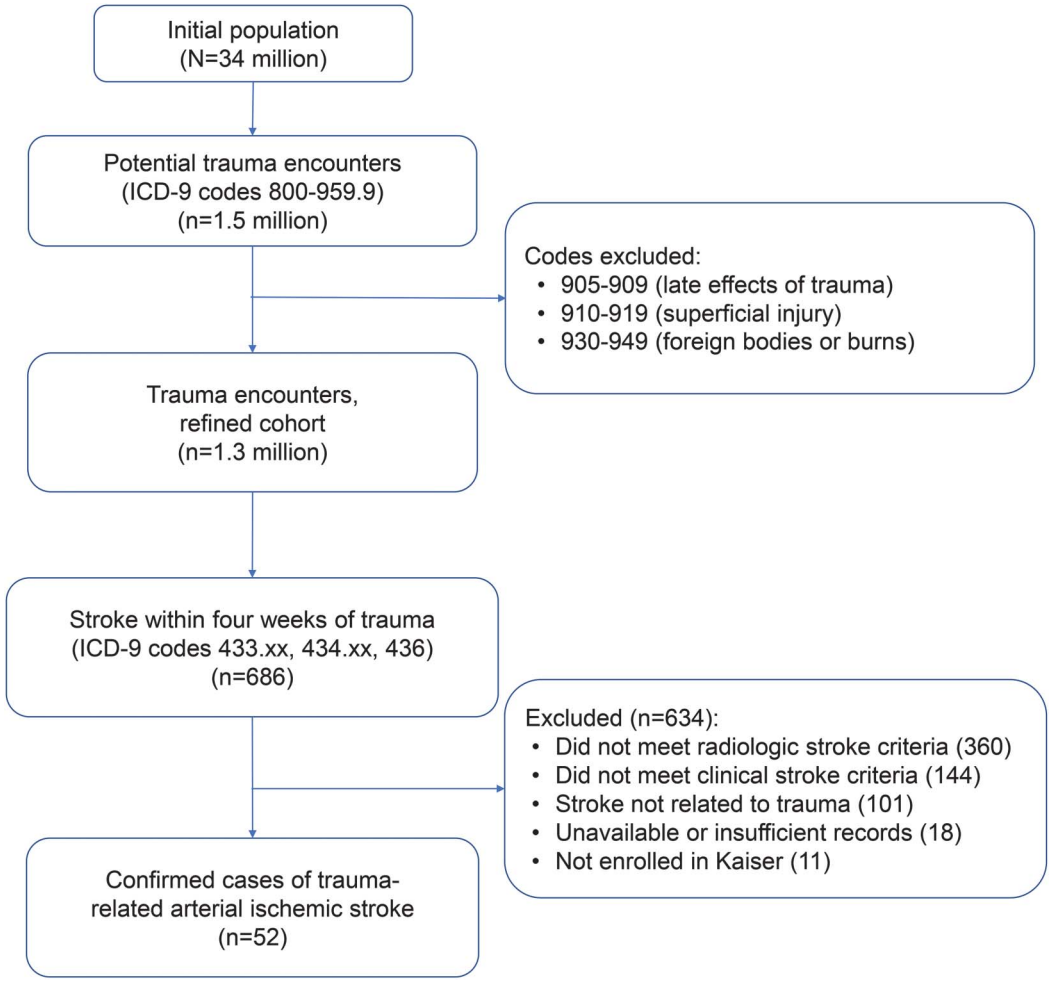
Population-based studies with systematic, longitudinal follow-up for stroke after trauma are lacking. From studies of BCVI, we hypothesized that the location of a traumatic injury (such as head, neck, chest, and back) is a primary risk factor for subsequent stroke. We sought to quantify the 4-week incidence, establish the timing, and stratify the risk of stroke by the location of traumatic injuries in a population of young patients evaluated in the emergency department or hospitalized for trauma.

METHODS

Study design and setting. We performed a retrospective, population-based study of arterial ischemic stroke attributed to trauma in children and young adults (<50 years of age) who were enrolled in Kaiser Permanente Northern California (KPNC) from 1997 through 2011. KPNC is an integrated health care delivery system comprising 21 nonrural community medical centers with extensive electronic and traditional medical records that can be accessed by Kaiser researchers with institutional review board approval. None of the KPNC medical centers is a level 1 trauma center, but the medical records of patients seen at the regional level 1 trauma centers are available and searchable through claims databases. Patient enrollment is generally representative of the socioeconomics and demographics of the regional population.

Standard protocol approvals, registrations, and patient consents. Institutional Review Boards at the University of California, San Francisco, and the Kaiser Foundation Research Institute, Northern California, approved the study, with a waiver of consent for minimal risk.

Figure 1 Flow chart of trauma cohort identification and stroke case confirmation



Children and young adults (<50 years of age) with ischemic stroke attributed to trauma while enrolled in Kaiser Permanente Northern California from 1997 through 2011 were identified electronically. A panel of neurologists adjudicated whether ischemic strokes were due to trauma after medical chart review. ICD-9 = *International Classification of Diseases-9*.

Cohort identification. We searched inpatient, outpatient, and claims databases to identify emergency encounters or hospital admissions of patients <50 years of age with trauma-related ICD-9 codes 800–959.9, excluding ICD-9 codes 905–909 (late effects of trauma), 910–919 (superficial injury), and 930–949 (foreign bodies and burns). We excluded encounters if the patient was a KPNC member for <4 weeks after the encounter. For our calculation of stroke incidence, individuals were allowed to contribute multiple trauma encounters if the encounters were >30 days apart, making the assumption that these could be considered independent. Repeat trauma encounters by a single individual were identifiable by a unique medical record number assigned to each KPNC enrollee.

Table 1 Characteristics of trauma-related stroke in 52 patients <50 years of age enrolled in a Northern California integrated health system, 1997–2011

	n (%)
Vascular imaging study after trauma (N = 28)	
CTA head or neck	19 (68)
MRA head or neck	14 (50)
DSA head or neck	8 (29)
Results, vascular imaging after trauma (N = 28)	
Arterial dissection	14 (50)
Penetrating vascular injury	4 (14)
Indeterminate injury	3 (11)
Normal	7 (25)
Stroke signs and symptoms	
Hemiparesis	29 (56)
Altered mental status	22 (42)
Change in speech	16 (31)
Headache	13 (25)
Change in gait	5 (10)
Seizure	3 (6)
Stroke imaging studies	
CT head	50 (96)
MRI brain	36 (69)
MRA head or neck	27 (52)
CTA head or neck	22 (42)
DSA head or neck	10 (19)
Results, vascular imaging after stroke (N = 41)	
Arterial dissection	21 (51)
Arterial occlusion	20 (49)
Arterial stenosis	12 (29)
Psuedoaneurysm	4 (10)
Normal	8 (20)
Infarct characteristics	
Single	38 (73)
Multiple	14 (27)
Large vessel	40 (77)
Small vessel/lacunar stroke	8 (15)
Indeterminate[a]	6 (12)
Infarct vascular territory	
Middle cerebral artery	31 (60)
Vertebrobasilar	17 (33)
Posterior cerebral artery	9 (17)
Anterior cerebral artery	3 (6)

Abbreviations: CTA = CT angiography; DSA = digital subtraction angiography; MRA = magnetic resonance angiography.
[a] Indeterminate if small or large vessel infarct.

Stroke ascertainment. From the trauma cohort, we identified possible arterial ischemic strokes within 4 weeks of the trauma through ICD-9 codes 433.xx, 434.xx, and 436[11] and cross-referencing a prior KPNC pediatric stroke study.[12] Because ICD-9 ischemic stroke codes may have limited accuracy,[13]particularly in young patients,[14,15] and because trauma patients may have nonischemic brain injuries that could be miscoded as an ischemic stroke, a neurologist reviewed medical records to confirm the strokes, using the following criteria: (1) a documented focal neurologic deficit of acute onset and (2) CT or MRI showing a focal brain arterial ischemic infarct in a location and of a maturity consistent with the neurologic signs and symptoms. Two neurologists independently reviewed records to determine if the stroke was attributable to trauma. A third neurologist adjudicated disagreements.

Control selection for case-control analysis. For each stroke case attributed to trauma, we randomly identified 3 stroke-free control encounters from the trauma cohort.

Data abstraction. A medical record analyst abstracted data regarding trauma and stroke using standardized protocols. Location of trauma to body regions was defined by documented injuries to head, neck, chest, back, abdomen, or extremities and were not mutually exclusive. Multisystem trauma was defined as documentation of "multisystem" or "multiorgan" trauma or documentation of trauma to multiple organs, head and organs, extremities and organs, or head and extremities. Cerebrovascular angiography included CT angiography (CTA), magnetic resonance angiography, or digital subtraction angiography of the head or neck. All data were reviewed by a neurologist for accuracy. Patients with insufficient records for data abstraction were excluded.

Statistical analysis. We used Stata 14 (StataCorp, College Station, TX). A kappa statistic was calculated for interrater agreement of stroke attributed to trauma. We described demographics and patient characteristics with summary statistics. We calculated stroke incidence as a proportion of trauma-related strokes per 100,000 trauma encounters. As a secondary analysis, we calculated stroke incidence by stroke ICD-9 codes to provide a comparison with studies using ICD-9 codes for the outcome. For trauma patients who had a stroke, we graphically demonstrated time to stroke using Kaplan-Meier failure curves, with trauma as the time of origin and stroke as the failure event, right-censoring at death or 4 weeks after trauma.

For the case-control study, we used logistic regression to calculate odds ratios (ORs) and 95% confidence intervals (CIs). To determine how the location of traumatic injuries (head, neck, chest, back, abdomen, or extremity) contributed to the magnitude of stroke risk, we first examined univariate models comparing patients with trauma in each anatomic

location to patients without trauma in that location. For example, patients with head injury were compared to patients without head injury to obtain an OR for the association of head injury to stroke. We then adjusted the estimated OR for demographics (sex, race, age) and markers of trauma severity (multisystem trauma, motor vehicle collision, arrival by ambulance, intubation). The case-control portion of our study consisted of only one trauma encounter per participant (both cases and controls).

RESULTS From 34 million children and young adults enrolled in KPNC over the 15-year study period, we identified 989,333 patients who had 1,308,009 trauma encounters and 4 weeks of follow-up. Only the top 1% contributed more than 4 trauma encounters. Within the cohort, 686 patients had an ischemic stroke ICD-9 code within 4 weeks of trauma. The 4-week incidence of stroke identified by ICD-9 codes is 52.5 (CI 48.6–56.5) per 100,000 trauma encounters (0.052%). After chart review, 153 patients met criteria for stroke and were reviewed for attribution of stroke to trauma. Seven of these required adjudication by a third neurologist to reconcile disagreements ($\kappa = 0.9$). After adjudication, we attributed 52 cases of arterial ischemic stroke to trauma (figure 1). The 4-week incidence of arterial ischemic stroke attributable to trauma is 4.0 (CI 3.0–5.2) strokes per 100,000 trauma encounters (0.004%).

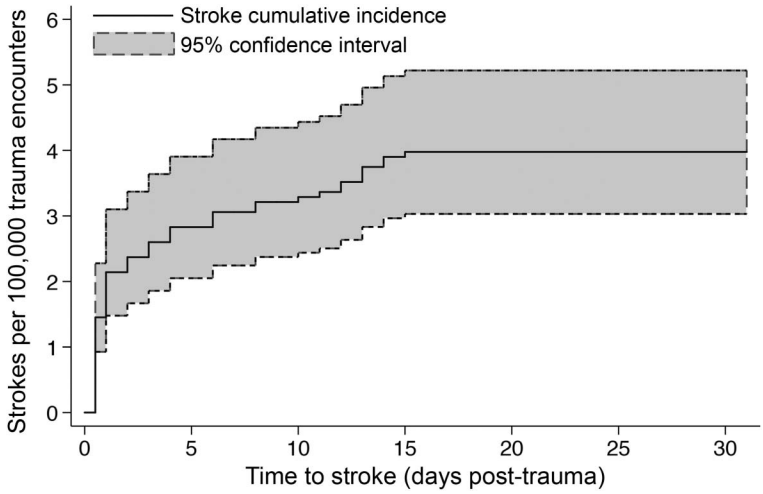
Cases of ischemic stroke related to trauma. Stroke and imaging characteristics of the 52 patients with stroke attributed to trauma are provided in table 1. In 19 patients (37%), the stroke occurred on the day of the trauma. All 52 strokes occurred by day 15 (figure 2). The most common location for traumatic injury was the head (in 69%), followed by injuries to the neck (31%) and chest (29%). Head injuries were associated with neck injuries ($p = 0.01$) but not injuries in other locations. In addition to trauma, other factors that could have contributed to stroke included septicemia in one patient, cocaine/amphetamine intoxication in one patient, and arrhythmia in one patient. A neurologic deficit at discharge was documented in 38 (73%).

All stroke patients had brain parenchymal imaging (MRI of the brain or CT of the head). Cerebrovascular angiography was performed in 28 (54%) as part of the trauma workup and in 41 (79%) as part of the stroke workup. In 7 (25%) of the 28 stroke patients who had screening cerebrovascular angiography for trauma, no arterial injury was detected. All 7 stroke patients with normal initial vascular imaging after trauma had repeat vascular imaging after the stroke occurred, documenting arterial stenosis or occlusion in 5 of the cases (table e-1 at Neurology.org).

Comparison of trauma in cases and controls. The case-control study compared the 52 stroke patients to 156 stroke-free controls (table 2). Among motor vehicle collisions in which speed was estimated in the medical record (13/24 cases and 8/15 controls), speed >35 miles per hour was associated with a 21-fold increase in stroke risk (OR 21.0, 95% CI 1.8–248.1). Medical acuity for trauma may have been higher for stroke patients compared to controls, with greater likelihood of presentation to care by ambulance, admission to the hospital, admission to the intensive care unit, and intubation compared to controls. Among stroke cases, there were 7 (13%) fatalities. The cause of death was attributed to stroke in 3 patients, head trauma in 3, and respiratory failure due to high cervical injury in 1. There were no trauma-related fatalities among controls.

Association of ischemic stroke with injury location. Multisystem trauma was highly associated with risk of ischemic stroke (table 2). Injuries to the head, neck, chest, back, and abdomen were each associated with increased risk of stroke in univariate analyses compared to patients without trauma in these locations. After adjusting for age, race, sex, and markers of trauma severity (multisystem trauma, motor vehicle collision, arrival by ambulance, intubation), an association with an increased stroke risk persisted for patients with injury to the head compared to those without head injury (OR 4.1, CI 1.1–14.9; $p = 0.031$) and patients with injury to the neck compared to those without neck injury (OR 5.6, CI 1.03–30.9; $p = 0.046$) (table 3).

Figure 2 Incidence of ischemic stroke after trauma



Kaplan-Meier failure curve demonstrates trauma-related arterial ischemic strokes among 1,308,009 medical encounters for trauma in children and young adults <50 years of age.

Table 2 Demographics and characteristics of 52 trauma patients with trauma-related ischemic stroke and 156 controls with no stroke after trauma

Characteristics	Cases, N = 52 (%)	Controls, N = 156 (%)	OR	95% CI	p Value
Age, y, median (IQR)[a]	34 (18-44)	24 (12-39)			0.109
40-50	20 (38)	36 (23)	Ref		
30-39	8 (15)	23 (15)	0.6	0.2-1.7	0.345
20-29	6 (12)	33 (21)	0.3	0.1-0.9	0.033
10-19	10 (19)	34 (22)	0.5	0.2-1.3	0.162
0-9	8 (15)	30 (19)	0.5	0.2-1.2	0.131
Male	34 (65)	92 (59)	1.3	0.7-2.5	0.413
Race/ethnicity					
White/Caucasian	25 (48)	58 (37)	Ref		
Latino/Hispanic	15 (29)	39 (25)	0.9	0.4-4.2	0.768
Black/African American	5 (10)	29 (19)	0.4	0.1-1.2	0.090
Asian/Pacific Islander	5 (10)	9 (6)	1.3	0.4-4.2	0.676
Other/Unknown	2 (4)	21 (13)	0.2	0.04-1.0	0.052
Multisystem trauma	26 (50)	13 (8)	11.0	5.0-24.1	<0.001
Trauma locations					
Head injury	36 (69)	26 (17)	11.3	5.5-23.2	<0.001
Neck injury	16 (31)	8 (5)	8.2	3.3-20.7	<0.001
Chest injury	15 (29)	4 (3)	15.4	4.8-49.1	<0.001
Back injury	10 (19)	11 (7)	3.1	1.2-7.9	0.015
Abdominal injury	10 (19)	3 (2)	12.1	3.2-46.1	<0.001
Extremity injury	11 (21)	83 (53)	0.2	0.1-0.5	<0.001
Mechanism of trauma					
Motor vehicle accident	24 (46)	15 (10)	8.1	3.8-17.3	<0.001
Fall	8 (15)	11 (7)	0.5	0.2-1.4	0.170
Sports-related injury	4 (8)	2 (1)	1.6	0.2-9.6	0.586
Self-inflicted	2 (4)	2 (1)	3.1	0.4-22.5	0.267
Assault	2 (4)	3 (2)	0.5	0.1-3.2	0.459
Arrived by ambulance	36 (69)	14 (9)	25.5	11.0-59.1	<0.001
Admitted to hospital	41 (79)	6 (4)	92.6	32.3-265.3	<0.001
Admitted to ICU	35 (67)	2 (1)	158.5	35.0-717.9	<0.001
Intubated	26 (50)	1 (1)	155.0	20.1-1192.1	<0.001

Abbreviations: CI = confidence interval; ICU = intensive care unit; IQR = interquartile range; OR = odds ratio; Ref = reference category.
ORs reflect univariate analyses.
[a] Equality of medians test.

Stroke risk stratified by head or neck injury. Because head and neck injuries often occur together and it can be clinically difficult to isolate one from the other, we created a composite category of patients with head or neck injury. Compared to trauma patients with no head or neck injury, an injury to the head or neck was associated with an increased stroke risk in an unadjusted analysis (OR 24.9, CI 10.3–60.4; $p < 0.001$) and in an analysis adjusted for age, race, sex, and markers of trauma severity (OR 13.5, CI 2.8–64.8; $p = 0.001$). The 4-week incidence of arterial ischemic stroke attributable to trauma among patients with a head or neck injury was 37.35 strokes per 100,000 trauma encounters (0.04%), but only 0.56 per 100,000 encounters among patients without a head or neck injury (0.0006%).

DISCUSSION Among young patients evaluated in the emergency department or hospital from 1997 to 2011, we measured a 0.004% 4-week incidence of stroke due to trauma, or 3.98 strokes per 100,000

Table 3 Association of ischemic stroke with location of injury after adjustment for age, race, sex, and markers of severity (multisystem trauma, motor vehicle collision, arrival by ambulance, intubation)

Trauma location	OR	95% CI	*p* Value
Head injury	4.1	1.1–14.9	0.031
Neck injury	5.6	1.03–30.9	0.046
Chest injury	1.6	0.2–13.2	0.659
Back injury	1.3	0.2–8.8	0.819
Abdominal injury	1.7	0.2–18.2	0.661
Extremity injury	0.1	0.01–0.3	0.001

Abbreviations: CI = confidence interval; OR = odds ratio.

trauma encounters. The clinical significance of the incidence rate may be magnified by the widespread nature of the exposure. In 2011, 24.4 million patients <50 years old were treated in US hospital emergency departments for nonfatal injuries.[16] Extrapolating from our incidence rate suggests that 971 young people had a trauma-related stroke in the United States that year.

Indirect indicators of trauma severity such as multisystem trauma, motor vehicle collision, and utilization of higher levels of care were associated with greater stroke risk in our study. Over the last 2 decades, investigators at level 1 trauma centers have debated the optimal protocol to define a high-risk group to screen for BCVI and intervene to decrease stroke risk.[3,17–20] The Western Trauma Association[4] and the Eastern Association for the Surgery of Trauma[5] suggest an extensive list of injuries that should prompt screening with cerebrovascular angiography. More recently, trauma groups have suggested even more liberal screening using CTA in all patients with blunt multisystem trauma[21] or with trauma that warrants spine or chest CT.[17,22]

As trauma centers are moving towards obtaining screening vascular imaging studies with greater frequency, our incidence rate should give pause to further evaluate these practices. Between 1% and 13% of patients evaluated at a trauma center meet criteria for CTA.[3,23,24] This will likely grow with the recent call for more liberal screening, but even if the narrower criteria are applied to the estimated 24 million trauma visits in the United States annually, then roughly 240,000–3,120,000 patients will receive a CTA to screen for BCVI when fewer than 1,000 will have a trauma-related stroke.

Widespread BCVI screening is costly and exposes young patients to ionizing radiation and contrast agents. Further, stroke occurs in patients with initially normal angiography. In our cohort, half of the stroke patients had head or neck vascular imaging as part of their trauma workup. Among these, 25% of the vascular imaging studies were reported to be normal. Therefore, trauma protocols that focus solely on BCVI detection may not be the most effective way to prevent stroke.

In prior case series, stroke has been reported most frequently during the first 3 days after a traumatic injury.[6–8] In contrast, our study found that 40% of the strokes occurred more than 3 days after the trauma index date, with a flattening of the stroke incidence rate at 2 weeks post-trauma. Our delineation of a discrete, higher risk window of time may support alternative strategies. For example, could a subset of patients be identified for a study to forgo vascular imaging and instead receive a 2-week course of aspirin?

The potential success of this type of strategy would rest on inclusion of high-risk patients while excluding patients with an unacceptably high risk of bleeding. We found that the patients at highest stroke risk were those with head or neck injuries. Because of the nature of this retrospective study, we were unable to reasonably assess how many of these patients would have been eligible for antiplatelet therapy prior to stroke onset. More work to identify a target population and determine if randomization to a short-term antiplatelet could be safe, feasible, and cost-effective is needed.

The strengths of our study are (1) the well-defined, population-based cohort, allowing reasonable generalizability to patients at community medical centers and level 1 trauma centers; (2) the large size of the trauma cohort, allowing estimates with reasonably narrow CIs; (3) the systematic longitudinal follow-up for stroke outcomes; (4) the confirmation of strokes after chart review; and (5) the adjudication by a physician panel of strokes attributable to trauma.

Our study also has limitations. (1) We may have missed stroke cases if no stroke ICD-9 code was recorded, brain imaging was not done, or clinical symptoms were not documented, resulting in an underestimation of stroke incidence. (2) We were not able to view and primarily analyze radiographic images, but instead had to rely on clinical radiology reports. (3) Although we carefully reviewed records to determine the relationship of stroke and trauma, we may have misclassified (and excluded) some strokes caused by a trauma, or attributed some strokes as related to trauma when they were not. (4) We made the assumption that multiple traumas experienced by the same person were independent if they did not occur within 30 days. Only a small percentage of the cohort had multiple trauma encounters; thus we believed this was reasonable.

In this population-based study of children and young adults, head or neck injuries increase the risk

of subsequent stroke by 4-fold and 6-fold, respectively, during the 2 weeks after a trauma. Because stroke onset is often delayed, some of these strokes might be preventable. However, in one-quarter of the stroke cases with vascular imaging at the time of the trauma, imaging did not detect cerebrovascular injury. Trauma protocols that focus solely on detection of BCVI may miss opportunities to prevent some trauma-related strokes. Collaboration between stroke neurologists and trauma investigators could result in additional strategies to minimize unnecessary imaging and improve primary stroke prevention.

AUTHOR CONTRIBUTIONS

Study conception and design: Drs. Fox, Vinson, Sidney, Dicker, and Fullerton. Acquisition of data: Drs. Fox, Numis, Sidney, and Fullerton. Analysis and interpretation of data: Drs. Fox, Hills, and Fullerton. Drafting of manuscript: Drs. Fox and Hills. Critical revision: Drs. Fox, Hills, Vinson, Numis, Dicker, Sidney, and Fullerton.

ACKNOWLEDGMENT

The authors thank Barbara Rowe, RN (Division of Research, Kaiser Permanente Northern California), for medical record abstraction, and Mike Sorel, MPH (Division of Research, Kaiser Permanente Northern California), for programming of electronic searches.

STUDY FUNDING

Supported by the American Heart Association (Established Investigator Award 12EIA9130050; H.J.F.) and by the NIH (awards K02 NS053883 and KL2TR000143; C.K.F.).

DISCLOSURE

The authors report no disclosures relevant to the manuscript. Go to Neurology.org for full disclosures.

Received March 6, 2017. Accepted in final form August 16, 2017.

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